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Pulse Oximetry and Arterial Oxygen Saturation during Cardiopulmonary Exercise Testing

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Abstract

Introduction/Purpose—Peripheral capillary oxygen saturation (SpO_2) is used as surrogate for arterial blood oxygen saturation. We studied the degree of discrepancy between SpO_2 and arterial oxygen (SaO_2) and identified parameters that may explain this difference.

Methods—We included patients who underwent cardiopulmonary exercise testing (CPET) at Cleveland Clinic. Pulse oximeters with forehead probes measured SpO_2 and arterial blood gas (ABG) samples provided the SaO_2 both at rest and peak exercise.

Results—We included 751 patients, 54 ± 16 years old with 53 % of female gender. Bland-Altman analysis revealed a bias of 3.8% with limits of agreement of 0.3 to 7.9% between SpO_2 and SaO_2 at rest. A total of 174 (23%) patients had SpO_2 >= 5% of SaO_2, and these individuals were older, current smokers with lower FEV1 and higher $PaCO_2$ and carboxyhemoglobin. At peak exercise (n=631), 75 (12%) SpO_2 values were lower than the SaO2 determinations reflecting difficulties in the SpO_2 measurement in some patients. The bias between SpO_2 and SaO_2 was 2.6% with limits of agreement between −2.9 to 8.1%. Values of SpO_2 >= 5% of SaO_2 (n=78, 12%) were associated with the significant resting variables plus lower heart rate, oxygen consumption

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and oxygen pulse. In multivariate analyses, carboxyhemoglobin remained significantly associated with the difference between SpO_2 and SaO_2 both at rest and peak exercise.

Conclusion—In the present study, pulse oximetry commonly underestimated the SaO_2. Increased carboxyhemoglobin levels are independently associated with the difference between SpO_2 and SaO_2, a finding particularly relevant in smokers.

Keywords

pulse oximetry; arterial blood gas; cardiopulmonary exercise test; forehead probe; arterial oxygenation

Introduction

Peripheral capillary oxygen saturation (SpO_2) is commonly measured by pulse oximetry, which provides an indirect measurement of arterial oxygenation (SaO_2) based on the differential absorption of light by oxygenated and deoxygenated blood during pulsatile blood flow (1). Pulse oximetry offers a non-invasive and rapid determination of SaO_2, particularly when the SaO_2 is above 75% (2). Arterial blood gas (ABG) analysis provides a direct measurement of the arterial partial pressure of oxygen (PaO_2) and SaO_2 among other important parameters that are used to assess ventilation and acid base status. However, ABG analysis requires more time and expense as well as an arterial puncture (3). Thus, SpO_2 is routinely used as surrogate for SaO_2.

Precision and accuracy of SpO_2 readings can be affected by technical problems inherent to the measuring device or inadequate interpretation of the data (4). Some sources of error can be modified (i.e. improper probe placement, motion artifact, nail polish, and stray light) while others cannot (i.e., presence of carboxyhemoglobin, methemoglobin, and skin pigmentation). In cases of poor perfusion, the accuracy of the reading can be optimized by changing the probe to a different location (e.g. finger, ear lobe or forehead), applying heat or a vasodilator cream, placing the hand below the level of the heart or trying a different sensor or pulse oximeter (5,6).

A good agreement between SpO_2 and the reference method of ABG analysis has been reported by some authors (7), while others reported an over- or underestimation of the SaO_2 (8,9,10). Some studies have pointed out that SpO_2 may not always be a reliable method to predict SaO_2 (11, 12). High venous pressure states can falsely lower SpO_2 values (3,13). Furthermore, decreased accuracy of SpO_2 has been described in hypoxemic (8), hemodynamically compromised (9,10), and critically ill (14) patients, in whom an accurate and reliable monitoring is of major importance. Increases in carboxyhemoglobin and on certain occasions methemoglobin can lead to falsely normal or high SpO_2 readings, despite a low SaO_2 (13). The source of error caused by these two dyshemoglobins can be mitigated by using co-oximetry, which requires an ABG.

Most studies investigating agreement between SpO_2 and SaO_2 have concentrated on critically ill inpatient populations (3,14), and studies in healthier, outpatient cohorts both at rest and during exercise are lacking. The purpose of our study is to examine the degree of

discrepancy between SpO_2 and SaO_2 both at rest and at maximum exercise and identify parameters that may explain this difference. We hypothesize that the discrepancy between SpO_2 and SaO_2 increases at maximal exercise and that several factors (particularly carboxyhemoglobin) can explain this difference. To test our hypothesis we retrospectively examined a cohort of patients undergoing cardiopulmonary exercise testing (CPET) at our institution, compared SpO_2 with SaO_2 both at baseline and at peak exercise during CPET, and tested whether variables collected during CPET can explain the differences between SpO_2 and SaO_2.

Methods

Study Population

The study was approved by the Institutional Review Board of the Cleveland Clinic (IRB # 15-1288). Data from 751 patients referred for CPET at the Respiratory Institute of the Cleveland Clinic, from January 2010 to January 2015, were retrospectively analyzed. Informed consent was waived.

Pulse Oximetry and Arterial Oxygenation Determinations

Our CPET protocol included the use of pulse oximeters (Nonin Avant 4000 system, Nonin, Plymouth, MI, USA) with forehead probes to measure SpO_2 both at baseline and during exercise. We routinely placed two pulse oximetry probes on the forehead to test accuracy and only recorded the most reliable determination based on the quality of the SpO_2 waveform. Forehead oximetry probes were held in place by an elastic band. Respiratory therapists recorded the SpO_2 measurement only when the waveform had a dicrotic notch and was synchronized with the heart rate observed in the electrocardiographic monitoring (15). SpO_2 at the time of ABG acquisition was recorded both at rest and peak exercise to avoid temporal variations. Measurements at rest were done in the sitting position after the patient relaxed for at least 10 minutes.

As part of our CPET protocol, SaO_2 levels were obtained from ABG samples taken from individual punctures of the radial artery (usually the left one) while patients sat on the bicycle. We did not place an arterial catheter. ABG determinations were performed with co-oximetry using the ABL800 FLEX Blood Gas Analyzer (Radiometer, Brønshøj, Denmark). ABG determinations were reported at 37 degree Celsius without correction by the core body temperature. Both SpO_2 and SaO_2 determinations were taken at rest (before exercise) and at maximum exercise (during the last 1-2 minute, just before stopping). Reasons for not been able to obtain an ABG at maximum exercise include: more than two unsuccessful attempts, transition into the recovery phase, and gasometric findings indicative of venous blood. In every patient, we recorded the amount of oxygen supplementation administered at the time of the measurements. If the patient required any degree of oxygen supplementation, either at rest or during activities, the CPET (including baseline and exercise determinations) was done with a fraction of inspired oxygen (FiO_2) of 30% using a bag reservoir.

Cardiopulmonary Exercise Testing (CPET) Protocol

CPET was ordered by pulmonary physicians based on established indications and done on an electrically braked cycle ergometer following recommendations by the American Thoracic Society and American College of Chest Physicians (16). Electrodes were placed on the skin to be able to obtain a 12-lead electrocardiogram. Blood pressure (BP) was recorded using a cuff placed on the arm opposite to the ABG site. An appropriate face mask (7450 SeriesV2 Mask, Hans Rudolph, Shawnee, KS, USA) size was selected and a good seal was verified. Measurements were obtained with the MedGraphics Ultima system (MGC Diagnostics, Saint Paul, MN, USA) following the manufacturer's recommendations. We used different maximal incremental exercise testing protocols based on the evaluation of each patient and with the intention of adjusting the exercise duration to approximately 8-12 minutes. Tests were terminated when the patient reached exhaustion (defined as 10/10 on the Borg scale or the inability to maintain pedal speed) or developed pronounced dizziness, leg, knee, chest or back pain (16).

For our study we recorded the following CPET variables: work rate, oxygen uptake (VO_2), carbon dioxide output (VCO_2), respiratory exchange ratio, anaerobic threshold, tidal volume, respiratory rate, minute ventilation (V_E), V_E/VCO_2, V_E/VO_2, breathing reserve, end-tidal PCO_2, heart rate, heart rate reserve, blood pressure, oxygen pulse, exercise duration and reasons for stopping the test. We also recorded SpO_2 and SaO_2 as well as other ABG results (partial pressure of oxygen in arterial blood (PaO_2), alveolar–arterial difference for oxygen pressure, $PaCO_2$, pH, bicarbonate, lactate, hemoglobin and carboxyhemoglobin), maximum voluntary ventilation measured at baseline, and forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), and FEV1/FVC (17) both at baseline and immediately after exercise.

Other variables

We collected data on age, gender, height, weight, smoking status, type of CPET protocol (5 to 30 Watts per minute), and reasons for the test. Reasons for ordering CPET were clustered in four groups including a) unexplained dyspnea, b) pre-operative work-up for cardiovascular or lung surgeries, c) diagnose or evaluate treatment response in exercise induced asthma, and d) others. In addition, we reviewed the patient's medications, time and contents of the last meal.

Statistical Analysis

Descriptive statistics (mean ± SD, median (interquartile range (IQR) or n (%)) were used to summarize demographic and clinically relevant variables. Continuous variables were compared with t-test. Categorical variables were tested using chi-square or Fisher exact test, when appropriate. Univariate linear regression was used to examine whether certain variables of interest eloquent were associated with the difference between SpO_2 and SaO_2 both at rest and peak exertion. Multivariate analysis was subsequently performed to examine the association between variables that achieved a p value < 0.10 in the univariate analysis. Variables in the models were tested for multicollinearity using variance inflation factors (VIF) and removing variables with a value larger than 4. Bland-Altman analysis was used to display the degree of bias and limits of agreement between SpO_2 and SaO_2 both at rest and

peak exertion, using SaO_2 as the gold standard in the x axis. We compared the patient characteristics at rest and during exercise between subjects that showed a difference between SpO_2 and SaO_2 < 5% or >= 5%. This prespecified but arbitrary cut-off was chosen to reflect an eloquent difference between measurements. Statistical significance was defined as a p-value less than 0.05. All analyses were performed using R: a Language and Environment for Statistical Computing (version 3.3.0).

Results

Patient Demographics

The mean age of the cohort (n=751) was 54.1 ± 16.1 years. A total of 355 (47%) patients were male. The majority of the tests were ordered for unexplained dyspnea (n=558 [74.3%]). Table 1 presents the baseline characteristics of the patients.

Oximetry at baseline

Results of the ABG analysis, spirometry and CPET at baseline are shown in Table 2. The baseline SpO_2 was 98.2 ± 1.8 %. The test was done on room air in 735 (97.9%) patients and on gas with 30% FiO_2 in the rest (n=16, 2.1%). The SpO_2 in individuals on room air and FiO_2 30% was 98.2 ± 1.8 and 98.3 ± 1.3%, respectively. Meanwhile, the SaO_2 was 94.4 ± 1.7 and 95.2± 1.3% for those breathing room air and FiO_2 of 30%, respectively. At rest, Bland-Altman analysis comparing SpO_2 with SaO_2 (gold standard) demonstrated a bias of 3.8% with limits of agreement of −0.3% to 7.9% (Figure 1, panel A). The vast majority (96.8%) of SpO_2 values were higher than the SaO_2 determinations and it appears that the lower the SaO_2 the more pronounced the gap with SpO_2 (Figure 1, panel A).

To better identify the characteristics associated with a larger difference between SpO_2 and SaO_2 at rest, we compared patients in whom the SpO_2 was >= 5% points higher than the SaO_2 (n=174, 23%) vs those with <5% points difference (n=577, 77%) (Table 3). We noted that when the difference between SpO_2 and SaO_2 was >= 5%, patients were older, with a higher proportion of current smokers, higher carboxyhemoglobin and $PaCO_2$ and lower FEV1, end-tidal CO_2 and hemoglobin (table 3).

In a multivariate analysis, we noted that the variables that were independently associated with the difference between SpO_2 and SaO_2 were current smoker status (β=1.92 for current vs never smokers and β=1.47 for current vs ex-smokers, p<0.001 for both), higher carboxyhemoglobin (per 1 % change, β=0.49, p<0.001), and lower PaO_2 (per 1 mmHg change, β=−0.125, p=0.02), FEV1 (per 1 L change, β=−1.15, p=0.01), maximal voluntary ventilation (per 1 L/min change, β=−0.009, p=0.02), and hemoglobin (per 1 g/dL change, β= −0.125, p=0.02). Similar results were noted when we excluded maximal voluntary ventilation (a variable with a VIF close to 4) from the model.

Oximetry at peak exercise

In 120 patients (16%) ABG was obtained at baseline but could not be obtained at peak exercise, therefore, at total 631 (84%) patients had both ABG and SpO_2 at peak exercise. Seventy-eight (12.4%) patients had a SpO_2 >= 5% points than the SaO_2. Bland-Altman

analysis showed a bias of 2.6% with limits of agreement of −2.9% and 8.1% (Figure 1 panel B). Interestingly, at peak exercise, several SpO_2 values (n= 75, 11.9%) were lower than the SaO_2 determinations. At peak exercise, a SpO_2 >= 5% point higher than the SaO_2 was associated with older age, lower height and weight, current smoker status, higher $PaCO_2$, bicarbonate, and carboxyhemoglobin, lower hemoglobin, FVC, FEV1, maximum heart rate, oxygen consumption, and O_2 pulse (Table 4). In multivariate analysis, the variables independently associated with the difference between SpO_2 and SaO_2 at peak exercise were lower FVC (per 1 L change, β=−0.27, p=0.001) and carboxyhemoglobin (per 1 % change, β=0.78, p<0.001).

A decrease in SaO_2 >= 4% during exercise is commonly considered clinically significant (16). At total of 26 (4%) patients had a decrease in SaO_2 >= 4% at maximum exercise and of them 20 (77%) had a drop in SpO_2 >= 4%. A drop in SpO_2 >= 4% had false positive and negative rates of 10% and 1%, respectively, for detecting a decrease in SaO_2 of >= 4%.

Discussion

The present study improves the understanding of the magnitude, direction and potential sources of error of pulse oximetry during exercise. It validates that carboxyhemoglobin is an important factor in explaining the difference between SpO_2 and SaO_2, and supports obtaining ABG determinations in certain patients, particularly current smokers. In addition we noted that a small proportion of patients had a falsely low SpO_2 reading during exercise that if not confirmed by ABG may lead to unnecessary testing.

The importance of pulse oximetry as a rapid, non-invasive tool to assess oxygenation cannot be understated. However, this methodology is susceptible to measuring error due to a variety of conditions. In general, the margin of error of SpO_2 is within 2% to 3% of the SaO_2 (14, 18, 19). Taking advantage of the large number of data collected and investigations performed at the time of CPET in our institution, we sought to test the accuracy of SpO_2 to estimate SaO_2 both at rest and at peak exercise with a particular focus in identifying factors accounting for the discrepancy between measurements.

In our laboratory and using the equipment described, we found that the bias between SpO_2 and SaO_2 did not increase during exercise (3.8% at baseline and 2.6% during the exercise), with the majority of the SpO_2 determinations overestimating SaO_2, particularly when the SaO_2 was below 90% or patients were current smokers. At peak exercise, the limit of agreement between SpO_2 and SaO_2 widened by one third from a gap of 8.25 to 10.99%, with more determinations in which SpO_2 was lower than SaO_2, reflecting limitations in the accurate determination of SpO_2, particularly during exercise. We identified several patient's characteristics associated with >= 5% points higher SpO_2 compared to SaO_2, such as older age, current smoker status, lower FEV1, and higher $PaCO_2$ and carboxyhemoglobin. In addition, during peak exercise, patients with a larger gap between SpO_2 and SaO_2 had lower heart rate, lower oxygen consumption and oxygen, pulse.

A few studies that included a small number of healthy patients tested the validity of pulse oximetry during maximal exercise and noted an under- (26,27) or overestimation (28,29) of

the SaO_2, with some suggesting a better precision when using a forehead sensor (26). To our knowledge, our study is the first to investigate the inconsistencies between SpO_2 measured with a forehead probe and SaO_2 in an outpatient cohort both at rest and peak exercise. The majority of our patients underwent CPET for unexplained dyspnea or as part of pre-operative work-up. Other studies have examined the discordance between SpO_2 and SaO_2; however, these cohorts consisted of critically ill inpatients or patients who recently underwent surgery (3, 8, 14, 20, 21, 22). Similar to these other investigations we noted that the bias between SpO_2 and SaO_2 at rest was 3.8%.

Motion artifact is one of the major causes of inaccurate pulse oximetry readings. We used a forehead probe to reduce the possibility of motion artifact and errors caused as a result of gripping the bicycle handlebars (15). We paid particular attention to the quality of SpO_2 waveform and test its accuracy against a second forehead probe. An accurate determination of SpO_2 is critical, since desaturations during CPET can lead to further invasive testing and expense. Although at rest we noted that SpO_2 systematically overestimates SaO_2, at peak exercise we observed that 10 (1.6%) patients had an SpO_2 below the SaO_2, in a range that could be considered hypoxemia (SpO_2 < 90%). In addition, in 10% of the patients we noted a drop in SaO_2 >= 4% during maximal exercise, at odds with the corresponding changes in SaO_2. These discrepancies could be related to forehead hypoperfusion, venous congestion with venous pulsations (30) or inaccuracies in the SpO_2 determination due to head motion during the activity. Given these findings and the additional information provided by the test, we recommend obtaining an ABG both at baseline and particularly at peak exercise during CPET (unless contraindicated) either using repeated punctures or an arterial line. ABG collection adds discomfort and an extra expense to patients. In addition, the ABG needs to be obtained at maximum exercise, since values can rapidly change in the recovery phase. Taking this caveats into consideration, we believe in confirming unexpected SpO_2 values with ABG analysis, particularly during exercise.

One of the main factors involved in the falsely high SpO_2 readings is carboxyhemoglobin (13), which absorbs light at approximately the same spectrum as oxyhemoglobin. Therefore, the determination presented by the pulse oximeter is a summation of oxyhemoglobin plus carboxyhemoglobin. This is particularly relevant in smokers, as noted in our analyses, and may explain the association with lower FEV1 and higher $PaCO_2$. In our cohort, carboxyhemoglobin was an important factor, however, it explained less than half of the difference between SpO_2 and SaO_2. Co-oximetry, which measures light absorbance at multiple wave lengths, is certainly needed if this source of error is suspected. In addition, data obtained at rest showed that PaO_2 and hemoglobin were inversely associated with the difference between SpO_2 and SaO_2; variables that may impact the accuracy of the pulse oximetry.

Our study is not without limitations: a) in 120 (16%) patients we could not obtain peak exercise ABG, b) pulse oximetry results were obtained with a forehead probe to minimize motion artifact (23), it is unclear if our findings can be extrapolated to finger probes, c) SpO_2 readings may vary according to the model of pulse oximeter used (13, 15, 25), and d) race was not recorded.

Conclusion

In the present study, pulse oximetry commonly underestimated the arterial oxygen saturation. Increased carboxyhemoglobin levels are major contributors to the difference between arterial and pulse oxygen saturation both at rest and peak exercise; a finding particularly relevant in smokers.

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The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation, and statement that results of the present study do not constitute endorsement by ACSM.

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References

1. Kacmarek RK, Stoller JK, Heuer AJ. Egan's Fundamentals of Respiratory Care. 10th. Mosby; 2012. Analysis and Monitoring of Gas Exchange; 398
2. Chapman KR, Liu FLW, Watson RM, Rebuck AS. Range of accuracy of two wavelength oximetry. Chest. 1986; 89:540–542. [PubMed: 3956279]
3. Zeserson E, Goodgame B, Hess JD, et al. Correlation of Venous Blood Gas and Pulse Oximetry With Arterial Blood Gas in the Undifferentiated Critically Ill Patient. J Intensive Care Med. 2016 E-publication ahead of print.
4. Kacmarek RK, Stoller JK, Heuer AJ. Egan's Fundamentals of Respiratory Care. 10th. Mosby; 2012. Analysis and Monitoring of Gas Exchange; 408
5. Kacmarek RK, Stoller JK, Heuer AJ. Egan's Fundamentals of Respiratory Care. 10th. Mosby; 2012. Analysis and Monitoring of Gas Exchange; 408–409.
6. Moyle JT. Uses and abuses of pulse oximetry. Arch Dis Child. 1996; 74(1):77–80. [PubMed: 8660057]
7. Bierman MI, Stein KL, Snyder JV. Pulse oximetry in the postoperative care of cardiac surgical patients. A randomized controlled trail. Chest. 1992; 102(5):1367–70. [PubMed: 1424853]
8. Benson JP, Venkatesh B, Patla V. Misleading information from pulse oximetry and the usefulness of continuous blood gas monitoring in a post cardiac surgery patient. Intensive Care Med. 1995; 21(5): 437–9. [PubMed: 7665754]
9. Vicenzi MN, Gombotz H, Krenn H, Dorn C, Rehak P. Transesophageal versus surface pulse oximetry in intensive care unit patients. Crit Care Med. 2000; 28(7):2268–70. [PubMed: 10921551]
10. Ibáñez J, Velasco J, Raurich JM. The accuracy of the Biox 3700 pulse oximeter in patients receiving vasoactive therapy. Intensive Care Med. 1991; 17(8):484–6. [PubMed: 1797894]
11. Jubran A, Tobin MJ. Reliability of pulse oximetry in titrating supplemental oxygen therapy in ventilator-dependent patients. Chest. 1990; 97(6):1420–5. [PubMed: 2347228]
12. Carter BG, Carlin JB, Tibballs J, Mead H, Hochmann M, Osborne A. Accuracy of two pulse oximeters at low arterial hemoglobin-oxygen saturation. Crit Care Med. 1998; 26(6):1128–33. [PubMed: 9635666]
13. Schnapp LM, Cohen NH. Pulse oximetry uses and abuses. Chest. 1990; 98(5):1244–1250. [PubMed: 2225973]
14. Van de louw A, Cracco C, Cerf C, et al. Accuracy of pulse oximetry in the intensive care unit. Intensive Care Med. 2001; 27(10):1606–13. [PubMed: 11685301]
15. Yamaya Y, Bogaard HJ, Wagner PD, Niizeki K, Hopkins SR. Validity of pulse oximetry during maximal exercise in normoxia, hypoxia, and hyperoxia. J Appl Physiol. 2002; 92(2):162–168. [PubMed: 11744656]
16. American Thoracic Society.; American College of Chest Physicians. ATS/ACCP Statement on cardiopulmonary exercise testing. Am J Respir Crit Care Med. 2003 Jan 15; 167(2):211–77. [PubMed: 12524257]

17. Miller MR, Hankinson J, Brusasco V, et al. ATS/ERS Task Force. Standardisation of spirometry. Eur Respir J. 2005 Aug; 26(2):319–38. [PubMed: 16055882]
18. Wouters PF, Gehring H, Meyfroidt G, et al. Accuracy of pulse oximeters: the European multi-center trial. Anesth Analg. 2002; 94(1 Suppl):S13–6. [PubMed: 11900030]
19. Webb RK, Ralston AC, Runciman WB. Potential errors in pulse oximetry. II. Effects of changes in saturation and signal quality. Anaesthesia. 1991; 46(3):207–12. [PubMed: 2014899]
20. Shibata O, Kawata K, Miura K, Shibata S, Terao Y, Sumikawa K. Discrepancy between SpO2 and SaO2 in a patient with severe anemia. J Anesth. 2002; 16(3):258–60. [PubMed: 14517653]
21. Ahmed S, Siddiqui AK, Sison CP, Shahid RK, Mattana J. Hemoglobin oxygen saturation discrepancy using various methods in patients with sickle cell vaso-occlusive painful crisis. Eur J Haematol. 2005; 74(4):309–14. [PubMed: 15777343]
22. Stewart KG, Rowbottom SJ. Inaccuracy of pulse oximetry in patients with severe tricuspid regurgitation. Anaesthesia. 1991; 46(8):668–70. [PubMed: 1887977]
23. Tomlinson AR, Levine BD, Babb TG. Low Pulse Oximetry Reading: Time for Action or Reflection? Chest. 2017; 151(4):735–736. [PubMed: 28390625]
24. Schnapp LM, Cohen NH. Pulse oximetry. Uses and abuses. Chest. 1990; 98(5):1244–50. [PubMed: 2225973]
25. Thilo EH, Andersen D, Wasserstein ML, Schmidt J, Luckey D. Saturation by pulse oximetry: comparison of the results obtained by instruments of different brands. J Pediatr. 1993; 122(4):620–6. [PubMed: 7681875]
26. Yamaya Y, Bogaard HJ, Wagner PD, Niizeki K, Hopkins SR. Validity of pulse oximetry during maximal exercise in normoxia, hypoxia, and hyperoxia. J Appl Physiol (1985). 2002; 92(1):162–168. [PubMed: 11744656]
27. Norton LH, Squires B, Craig NP, McLeay G, McGrath P, Norton KI. Accuracy of pulse oximetry during exercise stress testing. Int J Sports Med. 1992; 13(7):523–527. [PubMed: 1459747]
28. Wood RJ, Gore CJ, Hahn AG, et al. Accuracy of two pulse oximeters during maximal cycling exercise. Aust J Sci Med Sport. 1997; 29(2):47–50. [PubMed: 9242977]
29. Orenstein DM, Curtis SE, Nixon PA, Hartigan ER. Accuracy of three pulse oximeters during exercise and hypoxemia in patients with cystic fibrosis. Chest. 1993; 104(4):1187–1190. [PubMed: 8404189]
30. Sami HM, Kleinman BS, Lonchyna VA. Central venous pulsations associated with a falsely low oxygen saturation measured by pulse oximetry. J Clin Monit. 1991; 7(4):309–312. [PubMed: 1812874]

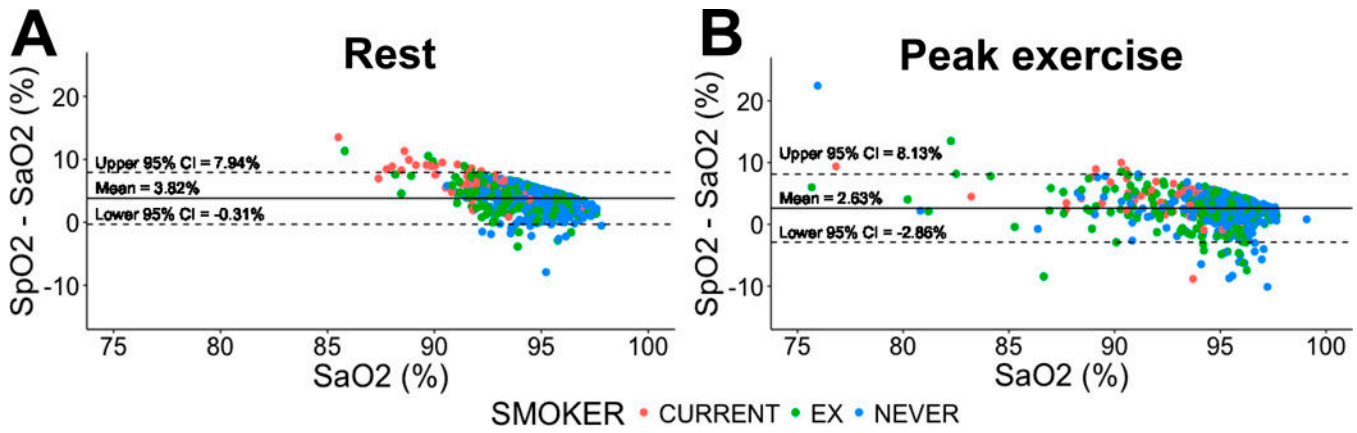

Figure 1. Bland-Altman plots testing the differences between SpO_2 and SaO_2 at rest and peak exercise

Plots show the mean difference and 95% confidence interval between SpO_2 and SaO_2, labeled by smoking status (current smoker, ex-smoker and never smokers). **Panel A:** determinations at rest. **Panel B:** determinations at peak exercise.

Table 1

Baseline Characteristics

Variable	Baseline determinations mean ± SD, n (%)
n	751
Age (years)	54 ± 16
Male gender	355 (47.3)
Height (cm)	172.0 ± 6.2
Weight (kg)	84.2 ± 21.6
BMI (kg/m^2)	29.0 ± 7.0
Smoking Status *	
Current Smoker	68 (9.1%)
Ex-Smoker	307 (41.0)
Never Smoker	374 (49.9)
Reason for CPET Testing ^	
Unexplained Dyspnea	558 (74.3)
Pre-Operative Workup	180 (24.0)
Exercise-Induced Asthma	3 (0.4)
Other	9 (1.2)

Abbreviations: BMI = body mass index; CPET = cardiopulmonary exercise testing; SD = standard deviation.

* Smoking status is missing in 2 patients,

^ reason for CPET testing is missing in 1 patient.

Table 2

CPET determinations at rest and peak exercise

Variable	Resting determinations mean ± SD, n (%)	Peak exercise determinations mean ± SD, n (%)
n	**751**	**631**
ABG Parameters		
pH	7.42 ± 0.03	7.35 ± 0.05
PaO_2 (mmHg)	87.8 ± 11.4	97.9 ± 16.9
$PaCO_2$ (mmHg)	37.7 ± 4.2	34.4 ± 5.9
HCO3 (meq/L)	24.2 ± 2.2	18.7 ± 4.9
SaO_2 (%)	94.4 ± 1.7	94.5 ± 2.8
Hemoglobin (g/dL)	13.9 ± 2.0	15.0 ± 4.0
Carboxyhemoglobin (%)	1.49 ± 0.87	1.23 ± 0.77
A-a gradient (mmHg)	12.8 ± 11.4	19.5 ± 4.0
$SpO_2 - SaO_2$ (%)	3.82 ± 2.06	2.63 ± 2.75
Spirometry Parameters		
FEV1 (L)	2.6 ± 0.9	2.71 ± 1.05
FEV1 (% of predicted)	83.8 ± 20.2	
FEV1 % change		−1.9 ± 21.7
FVC (L)	3.4 ± 1.1	3.40 ± 1.31
FVC (% of predicted)	86.0 ± 17.9	
FVC % change		−2.2 ± 21.6
Minute ventilation (L/min)	11.8 ± 4.1	70.0 ± 25.0
CPET Parameters		
Heart rate (bpm)	82 ± 16	141 ± 26
Respiratory rate (rpm)	17 ± 5	42 ± 10
SpO_2 (%)	98.2 ± 1.8	97.1 ± 3.2
Oxygen consumption (ml/min)	319 ± 89	1760 ± 632
Oxygen consumption/kg (ml/min/kg)	3.9 ± 0.9	21.6 ± 8.8
Oxygen pulse (ml/min/bpm)	4.0 ± 1.2	12.5 ± 4.0
End tidal CO_2 (mmHg)	33.2 ± 5.0	34.3 ± 6.6

Abbreviations: ABG = arterial blood gas**;** BMI = body mass index; CO_2 = carbon dioxide; CPET = cardiopulmonary exercise testing; FEV1 = forced expiratory volume in one second; FVC = forced vital capacity; HCO3 = bicarbonate; O_2 = oxygen; PaO_2 = partial pressure of oxygen; $PaCO_2$ = partial pressure of carbon dioxide; SaO_2 = arterial hemoglobin oxygen saturation; SpO_2 = pulse oximetry; SD = standard deviation. FEV1 and FVC % change refer to the percent variation post-exercise spirometry when compared to the resting determination.

Table 3

Patient characteristics based on a difference between SpO_2 and SaO_2 < 5% or >= 5% at rest.

Variables	$SpO_2 - SaO_2$ <5% mean ± SD, n (%)	$SpO_2 - SaO_2$>=5% mean ± SD, n (%)	P-value (t-test, chi-square)
n (%)	577 (76.8)	174 (23.2)	
Age (years)	53 ±17	59 ±13	*<0.001*
Male gender	271 (47.0)	84 (48.3)	0.84
Height (cm)	170.3 ±10.0	168.2 ±10.5	0.01
Weight (kg)	84.6 ±21.4	82.7 ±22.3	0.30
BMI (kg/m^2)	29 ±7.0	29 ±6.8	0.88
Smoking status			*<0.001*
Current smoker	20 (3.5)	48 (27.6)	
Ex-smoker	236 (41.0)	71 (40.8)	
Never smoker	319 (55)	55 (31.6)	
Reason for CPET testing			*<0.001*
Unexplained dyspnea	447 (77.6)	111(63.4)	
Pre-operative workup	118 (20.5)	62 (35.4)	
Exercise induced asthma	3 (0.5)	0 (0)	
Other	7 (1.2)	2 (1.1)	
FiO_2 (%)			0.48
21%	563 (97.6)	172 (98.8)	
30 %	14 (2.4)	2 (1.2)	
ABG Parameters			
pH	7.43 ±0.03	7.42 ±0.03	*0.018*
PaO_2 (mmHg)	90.0 ±10.9	80.6 ±10.1	*<0.001*
$PaCO_2$ (mmHg)	37.5 ±4.3	38.4 ±3.8	*0.011*
HCO3 (mEq/L)	24.1 ±2.2	24.3 ±1.9	0.29
SaO_2 (%)	94.9 ±1.3	92.7 ±1.8	*<0.001*
Hemoglobin (g/dL)	14.0 ±2.0	13.5 ±2.0	*0.014*
Carboxyhemoglobin (%)	1.31 ±0.46	2.07 ±1.50	*<0.001*
A-a gradient (mmHg)	11.2 ±10.6	18.0 ±12.4	*<0.001*
Spirometry Parameters			
FEV1 (L)	2.7 ±0.9	2.3 ±0.9	*<0.001*
FEV1 (% of predicted)	85.4 ±19.5	78.7 ±21.6	*<0.001*
FVC (L)	3.5 ±1.1	3.26 ±1.1	*0.01*
FVC (% of predicted)	86.3 ±17.8	84.7 ±18.5	0.27
Minute Ventilation (l/min)	12.0 ±4.4	12.0 ±3.2	0.7
CPET Parameters			
Heart Rate (bpm)	82 ±16	83 ±15	0.57

Variables	**SpO_2 − SaO_2 <5% mean ± SD, n (%)**	**SpO_2 − SaO_2>=5% mean ± SD, n (%)**	**P-value (t-test, chi-square)**
Respiratory rate (rpm)	17 ±5.2	18 ±4.5	0.11
SpO_2 (%)	97.9 ±1.9	99.1 ±1.1	*<0.001*
Oxygen consumption (ml/min)	321 ±91	312 ±84	0.24
Oxygen consumption/Kg (ml/min/kg)	3.9 ±0.9	3.9 ±0.9	0.91
Oxygen pulse (ml/min/bpm)	4.0 ±1.3	3.9 ±1.1	0.11
End-tidal CO_2 (mmHg)	33.4 ±4.9	32.4 ±5.3	*0.018*

Abbreviations: Δ = Difference between, ABG = arterial blood gas**;** BMI = body mass index; CO_2 = carbon dioxide; CPET = cardiopulmonary exercise testing; FEV1 = forced expiratory volume in one second; FiO_2= inspired fraction of oxygen; FVC = forced vital capacity; HCO3 = bicarbonate; O_2 = oxygen; PaO_2 = partial pressure of oxygen; $PaCO_2$ = partial pressure of carbon dioxide; SaO_2 = arterial hemoglobin oxygen saturation; SpO_2 = pulse oximetry; SD = standard deviation

Table 4

Patient characteristics based on a difference between SpO_2 and SaO_2 < 5% or >= 5% at peak exercise.

Variables	$SpO_2 - SaO_2$ <5% mean ± SD, n (%)	$SpO_2 - SaO_2$ >=5% mean ± SD, n (%)	P-value (t-test, chi-square)
n (%)	553 (87.6)	78 (12.4)	
Age (years)	54 ±16	61 ±14	*0.001*
Male gender	276 (49.9)	32 (41.0)	0.178
Height (cm)	170.7 ±10.0	166.1 ±9.3	*<0.001*
Weight (kg)	85.9 ±21.4	79.0 ±21.3	*0.007*
BMI (kg/m^2)	29.4 ±6.8	28.5 ±7.0	0.12
Smoking status			*<0.001*
Current smoker (%)	29 (5.3)	21 (26.9)	
Ex-smoker (%)	235 (42.6)	39 (50.0)	
Never smoker (%)	299 (52.2)	18 (23.1)	
Reason for CPET testing			
Unexplained dyspnea	424 (78.2)	49 (55.1)	
Pre-operative workup	110 (20.3)	38 (42.7)	
Exercise induced asthma	2 (0.4)	0 (0)	
Other	5 (0.9)	2 (2.2)	
FiO_2 (%)			1.00
21%	542 (98.0)	76 (97.4)	
30%	11 (2.0)	2 (2.6)	
ABG Parameters			
pH	7.35 ±0.05	7.35 ±0.05	0.494
PaO_2 (mmHg)	100.2 ±15.9	81.8 ±15.0	*<0.001*
$PaCO_2$ (mmHg)	34.0 ±5.8	36.8 ±5.7	*<0.001*
HCO3 (mEq/L)	18.5 (5.0	20.0 ±3.3	*0.011*
SaO_2 (%)	94.9 (2.2	91.4 ±4.0	*<0.001*
Hemoglobin (g/dL)	15.0 (4.3	14.0 ±2.0	0.026
Carboxyhemoglobin (%)	1.14 ±0.59	1.85 ±1.37	*<0.001*
A-a gradient (mmHg)	17.8 ±13.2	32.0 ±13.7	*<0.001*
Spirometry Parameters			
FEV1 (L)	2.7 ±1.0	2.1 ±1.1	*<0.001*
FEV1(% change)	-1.1 ±19.8	-8.0 ±31.8	*0.009*
FVC (L)	3.5 ±1.3	2.8 ±1.4	*0.002*
FVC (% change)	-1.3 ±19.6	-8.6 ±31.7	*0.005*
Minute ventilation (l/min)	71.0 ±25.0	62.0 ±22.4	*<0.001*
CPET Parameters			
Heart rate (bpm)	142 ±26	132 ±24	*<0.001*

Variables	**SpO_2 − SaO_2 <5% mean ± SD, n (%)**	**SpO_2 − SaO_2 >=5% mean ± SD, n (%)**	**P-value (t-test, chi-square)**
Respiratory rate (rpm)	42 ±10	42 ±10	0.98
SpO_2 (%)	97.0 ±3.2	97.8 ±3.0	*0.041*
Oxygen consumption (ml/min)	1813 ±629	1387 ±519	*<0.001*
Oxygen consumption/kg (ml/min/kg)	22.0 ±9.0	18.2 ±7.0	*<0.001*
Oxygen pulse (ml/min/bpm)	12.8 ±4.0	10.6 ±3.4	*<0.001*
End-tidal CO_2 (mmHg)	32.5 ±5.3	32.0 ±4.7	0.639

Abbreviations: ABG = arterial blood gas**;** BMI = body mass index; CO_2 = carbon dioxide; CPET = cardiopulmonary exercise testing; FEV1 = forced expiratory volume in one second; FiO_2= inspired fraction of oxygen; FVC = forced vital capacity; HCO3 = bicarbonate; O_2 = oxygen; PaO_2 = partial pressure of oxygen; $PaCO_2$ = partial pressure of carbon dioxide; SaO_2 = arterial hemoglobin oxygen saturation; SpO_2 = pulse oximetry; SD = standard deviation